

### **REMARKS/ARGUMENTS**

Prior to the present amendments, Claims 7-15 were pending in this application, and stood rejected on various grounds. Claims 7 and 14 have been amended, Claims 12, 13 and 15 have been canceled. All amendments are fully supported by the specification as originally filed, including claims that are now canceled. The amendments do not add new matter. Since the current amendments are believed to place the application in condition for allowance or, at least, present the claims in a better form for consideration on appeal, the entry of the requested claim amendments after final rejection is respectfully requested. All amendments were made without prejudice or disclaimer. Applicants specifically reserve the right to pursue all deleted subject matter in one or more continuing applications.

#### **Detailed Action**

Re.3 Figures 25-26 were objected to, since "Applicant has not affirmed whether the figures depict in situ hybridization or immunohistochemistry graphs." It is submitted that the description of Figure legends, as provided in Applicants' response of November 28, 2005, makes it clear that Figures 25 and 26 depict in situ hybridization. This fact is hereby specifically affirmed, which should obviate the present rejection.

Re. 6 Claims 7-15 were rejected under 35 U.S.C. 112, first paragraph, because the specification, "while being enabling for a method of reducing the progression of rheumatoid arthritis in mammal, comprising administering to said mammal an effective amount of an immunoadhesion comprising the extracellular domain sequence of the polypeptide of SEQ ID NO:32," allegedly does not provide enablement for a method of treating any inflammatory disorder in a mammal, using the various immunoadhesin constructs recited in the claims.

In explaining the rejection, the Examiner notes that "the exemplification is drawn to reduction of the progression of CIA [collagen-induced arthritis] that received the STIgMA fusion protein, in an assay that measure[s] anti-collagen type II antibody titer, radiographs, 5CT and histopathology methods (see example 25)," and finds that "[b]esides rheumatoid arthritis, the specification does not teach how to effectively treat any inflammatory disorder or reach any therapeutic endpoint in mammals by administering the therapeutic composition." The Examiner further notes that Kim et al. (or record) teaches that the Z39Ig protein (identical to StigMA) may

be involved in mediating phagocytosis and/or antigen presentation, and cites Walker (or record) for the notion that the function of STIgMA is unknown, and adds that the ligands of STIgMA are also unknown. From these findings, the Examiner concludes that the claims are not enabled within their full scope.

Without acquiescing to the Examiner's position, or the reasoning underlying the Examiner's position, the claims have been amended to be drawn to the treatment of rheumatoid arthritis using STIgMA-Ig immunoadhesins, comprising an extracellular domain sequence of the polypeptide of SEQ ID NO:32. This scope is clearly enabled by the experimental results set forth in Example 25, since, as the Examiner has acknowledged, the collagen-induced arthritis (CIA) is a well recognized animal model of rheumatoid arthritis.

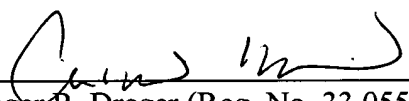
In view of the foregoing arguments and the current claim amendments, the Examiner is respectfully requested to reconsider and withdraw the present rejection.

All claims pending in this application are in prima facie condition for allowance, and an early action to that effect is respectfully solicited.

Please charge any additional fees, including any fees for additional extension of time, or credit overpayment to Deposit Account No. 08-1641 (referencing Attorney's Docket No. 39766-0100 CP1).

Respectfully submitted,

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